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Appetite regulation and energy balance

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Abstract
The decision to begin eating or to stop eating is a complex process. Hunger is primarily driven by hunger signals, like ghrelin and neuropeptide Y, originating from the gastrointestinal tract and from the hypothalamus. The hunger signals stimulate the seeking of food and the eating, being activating for the body and mind. Thirty minutes after the start of eating, satiety signals arise from the intestinal tract and, in between meals, from the adipose tissue and liver. Satiety signals are sedative and arrest the processing of food in the intestine, hence leading to termination of eating. One problem with overeating today is the ready access to palatable food, such as sucrose and fat. The palatable food works by weakening the satiety signals and activating the hunger signals. The reward system with endogenous opiates may also be activated.

Conclusions: Food and drinks rich in sucrose and fat should be given in a restricted way to children, since there is no biological control feedback to regulate the intake of such products.

Key Words: *Fat, sucrose, hunger, satiety, reward*

One of the key factors in the long-term success of a weight-reducing programme is the ability to maintain satiety in a context of reduced caloric intake. In fact, hunger is a significant predictor of weight gain after weight loss induced by an energy-restricted diet. Thus, hunger and satiety are crucial factors in determining energy balance. The regulation of food intake is based on a network of interactions forming a biological system, which is influenced by environment, i.e. the availability of nutrients and various psychological factors such as stress, as well as by a genetic predisposition. It is a complex system, as has been described in a number of recent reviews [1,2]. Basically food intake is controlled by excitatory and inhibitory signalling systems. The signals are generated both from the peripheral organs, like the intestine, the liver and the adipose tissue, and from the brain itself. The final targeting of the signals resides in the brain, in various nuclei of the hypothalamus. The identification of hunger and satiety peptides as well as their receptors in recent years has greatly increased our understanding of appetite regulation. Tools are continuously being targeted that could be useful in the pharmacological treatment of eating disorders; both anorexia and obesity.

Hunger signals and the drive to eat

The identification of appetite-regulating signals started with the characterization of powerful satiety peptides, focusing the attention of appetite control to satiety and the termination of a meal. It has since become clear that the drive for seeking food and eating is the central event in appetite regulation; satiety signals having the role of inhibiting hunger signals, rather than creating new signalling pathways. A couple of hunger peptides have been identified; the most important being neuropeptide Y (NPY), orexin and ghrelin. Ghrelin is produced in the stomach by endocrine cells, while NPY and orexin are produced in the hypothalamus of the brain. The hunger signals stimulate food intake and are upregulated during situations of increased energy demand, such as after fasting, after physical activity and during lactation. The hunger peptides also have an awakening effect, stimulating the seeking and collection of food. What determines the actual start of eating is still a matter of controversy. It has been suggested that a dip in blood glucose would be such a signal. Another start signal could be the release and secretion of digestive enzymes, which is elevated in obese subjects. It is important to note that the rise of hunger signals is not enough for the onset of feeding. Patients

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with anorexia nervosa have elevated levels of NPY, yet do not eat. The hunger peptides also have important effects on energy metabolism, by diverting the energy to the adipose tissue, at the same time restricting the flow of energy to skeletal muscle. Hence, the fat mass/fat-free mass ratio is significantly increased by the hunger peptides, in particular by NPY and ghrelin.

Satiety signals dampen hunger

Satiety signals are produced in the gastrointestinal tract, in the liver and in the adipose tissue. The gastrointestinal signals provide information on the fullness of the stomach as well as on the composition of the diet [3]. The message is taken to the brain through vagal primary afferent nerves, which terminate in the brain stem in the nucleus tractus solitarius, from there communicating with other hypothalamic nuclei affecting feeding behaviour. Examples of gastrointestinal signals are cholecystokinin, glucagon-like peptide 1, amylin, enterostatin and PYY. The liver transmits satiety signals to the brain mainly through energy metabolites derived from fat and glucose and through the level of ATP in the hepatocytes. The signalling to the brain occurs through afferent vagal pathways [4]. The adipose tissue controls the long-term regulation of appetite. This occurs through the concerted action of leptin and insulin, providing negative feedback signals for food intake in relation to body energy stores such as the adipose tissue [1]. Leptin and insulin both act centrally to regulate food intake, for which reason they need to be transported through the circulating blood and into the brain to reach central receptors. The leptin receptor is localized in the hypothalamus, including the arcuate nucleus and the PVN nucleus. The main effect of leptin is in the regulation of genes involved in feeding behaviour. Leptin reduces the expression of various hunger peptides, at the same time raising the expression of satiety peptides. The leptin signalling system has the potential to prevent obesity, yet it lacks effectiveness in all forms of obesity, since leptin secretion increases as the adipose tissue expands. This implies a form of leptin resistance, which could occur in the passage of leptin through the blood–brain barrier, as documented in obese subjects [5]. Insulin inhibits food intake when acting through central receptors. The inability of insulin to control body weight in obese subjects may also be a consequence of reduced passage through the blood–brain barrier.

In understanding the appetite regulation and energy balance of today, the influence of palatable food and its effects are of critical importance. Palatable food activates the reward system in a highly potent way, using pathways similar to alcohol and nicotine, by activation of the opioid system. The palatability of food has introduced the terms "food addiction" and "craving for food", which cause uncontrolled stimulation of appetite [6]. In this way, sucrose and sweet-tasting food have been shown to be efficient in stimulating food intake, in particular when present in sweet soft drinks. The highly increased prevalence of sweet drink consumption in children is an important cause of obesity [7]. Thus, it seems that the biology of appetite regulation has not been constructed for such high exposure to palatable food, explaining the epidemic of obesity occurring today in all societies. Eating is as much a means of achieving energy balance as it is a form of pleasure and reward. The drive to eat is one of the most powerful urges of animal and human behaviour. Indeed, it is a challenge to understand and regulate this behaviour!

Conclusion

Appetite regulation is governed by hunger and satiety signals released according to our state of energy balance: hunger signals in response to fasting, and satiety signals in response to feeding. Hunger signals drive us to seek and eat food, whereas satiety signals induce a state of non-stress and rest. There are two problems with appetite regulation today. One is the highly tasty food that overrides satiety signals and triggers the reward system. The second is a lack of physical activity, which makes overeating easier.

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